Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	15382	autologous	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L2	159235	bone marrow	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L3	738499	stromal cells	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L4	36216	(bone marrow) SAME (stromal cells)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L5	2704	autologous WITH (bone marrow)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L6	7288	autologous and ((bone marrow) SAME (stromal cells))	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L7	650261	GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L8	5400	(autologous and ((bone marrow) SAME (stromal cells))) and (GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L9	6295	angiogen? or neovascularization or "collateral blood vessel"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34

L10	877	((autologous and ((bone marrow) SAME (stromal cells))) and (GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase")) and (angiogen? or neovascularization or "collateral blood vessel")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L11	1	impaired WITH "autologous bone marrow"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L12	327	impaired WITH "bone marrow"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L13	25	(((autologous and ((bone marrow) SAME (stromal cells))) and (GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase")) and (angiogen? or neovascularization or "collateral blood vessel")) and (impaired WITH "bone marrow")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L14	1734	(autologous WITH (bone marrow)) and (GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L15	150	((autologous WITH (bone marrow)) and (GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase")) and (angiogen? or neovascularization or "collateral blood vessel")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34

L16	11	(((autologous WITH (bone marrow)) and (GM-CSF or GMCSF or HIF1 or HIF-1 or MCP1 or MCP-1 or EPAS1 or EPAS-1 or FGF or VEGF or PR39 or NOS or "fibroblas growth factor" or "nitric oxide synthase")) and (angiogen? or neovascularization or "collateral blood vessel")) and (impaired WITH "bone marrow")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L17	14	"WO 99/03973"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L18	2	"6063627".pn.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:34
L19	2458	"ex vivo" SAME (adenovirus or adenoviral or "Ad vector")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:36
L20	940	l19 and (angiogenesis or neovascularization or "collateral blood vessel" or arteriogenesis)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:37
L21	836	l20 and l2	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:37
L22	826	l21 and l7	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:37
L23	383	l22 and hypoxia	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:37
L24	187	l23 and ("early attaching" or CD34 or "CD-34")	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:39
L25	8	l24 and l5	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2004/12/02 13:39

	U	Document ID	Title
1	X	US 20040161421 A1	Intramyocardial injection of autologous bone marrow
2	X	US 20040131601 A1	Injection of bone marrow-derived cells and medium for angiogenesis
3	X	US 20040034196 A1	98 human secreted proteins
4	X	US 20040018969 A1	Nucleic acids, proteins, and antibodies
5	X	US 20030040088 A1	Secreted protein HT5GJ57
6	X	US 20030017500 A1	Secreted protein HT5GJ57
7	X	US 6534631 B1	Secreted protein HT5GJ57
8	X	US 6476195 B1	Secreted protein HNFGF20

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FILE 'MEDLINE, EMBASE, BIOSIS' ENTERED AT 13:41:43 ON 02 DEC 2004
L1         20136 S AUTOLOGOUS (S) "BONE MARROW"
L2          8319 S MARROW (S) "STROMAL CELLS"
L3        278021 S GM-CSF OR HIF-1 OR MCP-1 OR EPAS1 OR NOS OR FGF OR VEGF OR PR
L4            15 S L1 AND L2 AND L3
L5            10 DUP REM L4 (5 DUPLICATES REMOVED)
L6        104450 S ANGIOGEN? OR "COLLATERAL BLOOD VESSEL" OR NEOVASCULARIZATION
L7            10 S L1 AND L5
L8             3 S L7 NOT PY>=2001
L9        403646 S "BONE MARROW"
L10           75 S "IMPAIRED BONE MARROW"
L11         1545 S HIF1 OR EPAS1 OR GMCSF OR MCP1
L12        47967 S "EX VIVO"
L13        76046 S "GENE THERAPY" OR ("GENE TRANSFER" (P) THERAPY)
L14        78449 S ADENOVIRUS OR ADENOVIRAL OR "AD VECTOR"
L15       229568 S "VIRAL RELATED TOXICITY" OR IMMUNOTOXICITY OR "IMMUNE RESPONS
L16          799 S L12 AND L13 AND L14
L17           16 S L16 AND L1
L18            6 S L17 NOT PY>=2001
L19            4 DUP REM L18 (2 DUPLICATES REMOVED)
L20            1 S L16 AND L11
L21           40 S L16 AND L6
L22            5 S L21 AND L9
L23            4 DUP REM L22 (1 DUPLICATE REMOVED)
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ANSWER 1 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 1999355075 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10428121
TITLE: The effect of regional **gene therapy** with bone morphogenetic protein-2-producing bone-marrow cells on the repair of segmental femoral defects in rats.
AUTHOR: Lieberman J R; Daluiski A; Stevenson S; Wu L; McAllister P; Lee Y P; Kabo J M; Finerman G A; Berk A J; Witte O N
CORPORATE SOURCE: Department of Orthopaedic Surgery, University of California at Los Angeles, 90095, USA.
CONTRACT NUMBER: K11 AR01931-02 (NIAMS)
SOURCE: Journal of bone and joint surgery. American volume, (1999 Jul) 81 (7) 905-17.
Journal code: 0014030. ISSN: 0021-9355.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199908
ENTRY DATE: Entered STN: 19990820
Last Updated on STN: 19990820
Entered Medline: 19990809

AB BACKGROUND: Recombinant human bone morphogenetic proteins (rhBMPs) can induce bone formation, but the inability to identify an ideal delivery system limits their clinical application. We used **ex vivo adenoviral** gene transfer to create BMP-2-producing bone-marrow cells, which allow delivery of the BMP-2 to a specific anatomical site. The **autologous** BMP-2-producing **bone-marrow** cells then were used to heal a critical-sized femoral segmental defect in syngeneic rats. METHODS: Femoral defects in five groups of rats were filled with 5 x 10(6) BMP-2-producing bone-marrow cells, created through **adenoviral** gene transfer (twenty-four femora, Group I); twenty micrograms of rhBMP-2 (sixteen femora, Group II); 5 x 10(6) beta-galactosidase-producing rat-bone-marrow cells, created through **adenoviral** gene transfer of the lacZ gene (twelve femora, Group III); 5 x 10(6) uninfected rat-bone-marrow cells (ten femora, Group IV); or guanidine hydrochloride-extracted demineralized bone matrix only (ten femora, Group V). Guanidine hydrochloride-extracted demineralized bone matrix served as a substrate in all experimental groups. Specimens that were removed two months postoperatively underwent histological and histomorphometric analysis as well as biomechanical testing. RESULTS: Twenty-two of the twenty-four defects in Group I (BMP-2-producing bone-marrow cells) and all sixteen defects in Group II (rhBMP-2) had healed radiographically at two months postoperatively compared with only one of the thirty-two defects in the three control groups (beta-galactosidase-producing rat-bone-marrow cells, uninfected rat-bone-marrow cells, and guanidine hydrochloride-extracted demineralized bone matrix alone). Histological analysis of the specimens revealed that defects that had received BMP-2-producing bone-marrow cells (Group I) were filled with coarse trabecular bone at two months postoperatively, whereas in those that had received rhBMP-2 (Group II) the bone was thin and lace-like. Defects that had been treated with bone-marrow cells producing beta-galactosidase (Group III), uninfected bone-marrow cells (Group IV), or guanidine hydrochloride-extracted demineralized bone matrix only (Group V) demonstrated little or no bone formation. Histomorphometric analysis revealed a significantly greater total area of bone formation in the defects treated with the BMP-2-producing bone-marrow cells than in those treated with the rhBMP-2 ($p = 0.036$). Biomechanical testing demonstrated no significant differences, with the numbers available, between the healed femora that had received BMP-2-producing bone-marrow cells and the untreated (control) femora with respect to ultimate torque to failure or energy to failure. CONCLUSIONS: This study demonstrated that

BMP-2-producing bone-marrow cells created by means of **adenoviral** gene transfer produce sufficient protein to heal a segmental femoral defect. We also established the feasibility of **ex vivo** gene transfer with the use of biologically acute **autologous** short-term cultures of **bone-marrow** cells.

L19 ANSWER 2 OF 4 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN

ACCESSION NUMBER: 1998178569 EMBASE

TITLE: High efficiency myogenic conversion of human fibroblasts by **adenoviral** vector-mediated MyoD **gene transfer**. An alternative strategy for **ex vivo gene therapy** of primary myopathies.

AUTHOR: Lattanzi L.; Salvatori G.; Coletta M.; Sonnino C.; De Angelis M.G.C.; Gioglio L.; Murry C.E.; Kelly R.; Ferrari G.; Molinaro M.; Crescenzi M.; Mavilio F.; Cossu G.

CORPORATE SOURCE: G. Cossu, Dipto. di Istol. ed Embiologia Med., Universita di Roma La Sapienza, Via A. Scarpa, 14, 00100 Roma, Italy. cossu@axrma.uniroma1.it

SOURCE: Journal of Clinical Investigation, (15 May 1998) 101/10 (2119-2128).
Refs: 39
ISSN: 0021-9738 CODEN: JCINAO

COUNTRY: United States

DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 021 Developmental Biology and Teratology
022 Human Genetics

LANGUAGE: English

SUMMARY LANGUAGE: English

AB **Ex vivo gene therapy** of primary myopathies, based on **autologous** transplantation of genetically modified myogenic cells, is seriously limited by the number of primary myogenic cells that can be isolated, expanded, transduced, and reimplanted into the patient's muscles. We explored the possibility of using the MyoD gene to induce myogenic conversion of nonmuscle, primary cells in a quantitatively relevant fashion. Primary human and murine fibroblasts from skin, muscle, or **bone marrow** were infected by an E1-deleted **adenoviral** vector carrying a retroviral long terminal repeat- promoted MyoD cDNA. Expression of MyoD caused irreversible withdrawal from the cell cycle and myogenic differentiation in the majority (from 60 to 90%) of cultured fibroblasts, as defined by activation of muscle-specific genes, fusion into contractile myotubes, and appearance of ultrastructurally normal sarcomagenesis in culture. 24 h after **adenoviral** exposure, MyoD-converted cultures were injected into regenerating muscle of immunodeficient (severe combined immunodeficiency/beige) mice, where they gave rise to β-galactosidase positive, centrally nucleated fibers expressing human myosin heavy chains. Fibers originating from converted fibroblasts were indistinguishable from those obtained by injection of control cultures of lacZ-transduced satellite cells. MyoD-converted murine fibroblasts participated to muscle regeneration also in immunocompetent, syngeneic mice. Although antibodies from these mice bound to **adenoviral** infected cells in vitro, no inflammatory infiltrate was present in the graft site throughout the 3-wk study period. These data support the feasibility of an alternative approach to **gene therapy** of primary myopathies, based on implantation of large numbers of genetically modified primary fibroblasts massively converted to myogenesis by **adenoviral** delivery of MyoD **ex vivo**.

L19 ANSWER 3 OF 4 MEDLINE on STN DUPLICATE 2

ACCESSION NUMBER: 97169876 MEDLINE

DOCUMENT NUMBER: PubMed ID: 9017419
TITLE: A novel **gene therapy** strategy for elimination of prostate carcinoma cells from human bone marrow.
AUTHOR: Kim M; Wright M; Deshane J; Accavitti M A; Tilden A; Saleh M; Vaughan W P; Carabasi M H; Rogers M D; Hockett R D Jr; Grizzle W E; Curiel D T
CORPORATE SOURCE: Gene Therapy Program, University of Alabama at Birmingham 35294, USA.
CONTRACT NUMBER: 1RO1CA725332-01 (NCI)
SOURCE: Human gene therapy, (1997 Jan 20) 8 (2) 157-70.
Journal code: 9008950. ISSN: 1043-0342.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970612
Last Updated on STN: 20000303
Entered Medline: 19970603

AB We report a novel means to purge bone marrow of a specific subset of prostate carcinoma cells based on transductional and genetic selectivity. Using both **adenovirus**-polylysine-DNA complexes and E1A/B-deleted replication-deficient adenoviruses, we have demonstrated a transductional preference of these vectors for the prostate carcinoma cell lines DU 145, LNCaP, and PC-3 over primary human bone marrow cells and the leukemia cell line KG-1. We have also shown a genetic selectivity of an anti-erbB-2 intracellular single-chain antibody (sFv) encoding **adenovirus**, Ad21, for the erbB-2-positive prostate carcinoma cell lines DU 145 and LNCaP. Delivery of Ad21 resulted in cytotoxicity to the DU 145 and LNCaP, but not PC-3, cell lines and reduced the clonogenic capacity of DU 145 cells cultured alone or mixed with various ratios of irradiated human bone marrow. Finally, quantitative, competitive reverse transcription polymerase chain reaction (QC-RT-PCR) analysis demonstrated that Ad21 could effectively reduce DU 145 and erbB-2-positive primary prostate tumor contamination in bone marrow cultures. Delivery of Ad21 had no effect on the ability of progenitor cells to form colonies. These results suggest that an anti-erbB-2 sFv-encoding **adenoviral** vector is efficacious for removal of erbB-2-positive prostate carcinoma cells from human **bone marrow**, and demonstrates a novel method for **ex vivo** genetic purge of malignant cells from **bone marrow** for **autologous bone marrow** transplantation (ABMT) therapy.

L19 ANSWER 4 OF 4 on STN
ACCESSION NUMBER: 97019121 EMBASE
DOCUMENT NUMBER: 1997019121
TITLE: **Gene therapy** for malignant neoplasms of the CNS.
AUTHOR: Culver K.W.
CORPORATE SOURCE: K.W. Culver, Gene Therapy Res. Clinical Affairs, OncorPharm, 200 Perry Parkway, Gaithersburg, MD 20877, United States
SOURCE: Bone Marrow Transplantation, (1996) 18/SUPPL. 3 (S6-S9).
Refs: 20
ISSN: 0268-3369 CODEN: BMTRE
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article
FILE SEGMENT: 008 Neurology and Neurosurgery
016 Cancer
022 Human Genetics

025 Hematology
037 Drug Literature Index
038 Adverse Reactions Titles

LANGUAGE: English
SUMMARY LANGUAGE: English

AB Five different gene transfer protocols have progressed into human clinical trials for the treatment of brain tumors. Two utilize the in vivo transfer of the Herpes Simplex-thymidine kinase (HS-tk) gene by either retroviral or **adenoviral** gene transfer. HS-tk confers a sensitivity to the anti-herpes drug ganciclovir (GCV). Insertion of HS-tk into tumors and subsequent treatment with GCV has successfully eliminated tumors in experimental animal models despite less than a 100% gene transfer efficiency. This phenomenon, the 'bystander effect', allows the destruction of neighboring tumor cells not transduced with HS-tk. Two other approaches use **ex vivo** gene transfer of either the IL-2 or antisense insulin-like growth factor type 1 (IGF-1) genes into **autologous** tumor cells. In animal models, tumor cells genetically altered with antisense IGF-1 or IL-2 genes induce a potent cell-mediated antitumor response. The fifth approach uses the genetic modification of hematopoietic stem cells instead of tumor cells. In this approach, the multiple drug resistance (MDR-1) gene is transferred into stem cells to protect them from the toxic effects of certain chemotherapy drugs. This may allow the administration of higher doses without increasing **bone marrow** toxicity. Together, these clinical trials will provide critical information needed to develop improved gene transfer technologies for humans and to attain clinical benefit for cancer patients.

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